Upsala Journal of Medical Sciences. 2014; 119: 294

informa
healthcare

LETTER

Author's reply: Predictors of aspirin resistance

TAYFUR TOPTAS[1], EYYUP TASDEMIR[2], CENGIZ DEMIR[3], RAMAZAN ESEN[3] & MURAT ATMACA[4]

[1]*Department of Hematology, Van Training and Research Hospital, Van, Turkey,* [2]*Department of Internal Medicine, Van Training and Research Hospital, Van, Turkey,* [3]*Deparment of Hematology, Yuzuncu Yil University, Van, Turkey, and* [4]*Department of Endocrinology, Yuzuncu Yil University, Van, Turkey*

Yalcinkaya and Celik comment on the issue of the impact of aspirin esterase on aspirin resistance (1). It is probable that previous observations regarding hydrolysis of aspirin could be an underlying alternative explanation, which supports the relationship between high total cholesterol levels and aspirin resistance in our study.

The authors raise concerns about the relationship between inflammation and aspirin resistance. In contrast to what is claimed, in our study diabetic and non-diabetic subjects were comparable in terms of inflammatory conditions mentioned by Yalcinkaya and Celik such as hypertension (79.6% versus 70.3%, respectively, $p = 0.26$, data not shown in article), coronary heart disease (34.4% versus 32.4%, respectively, $p = 0.83$), and American Heart Association/American College of Cardiology stage C or D chronic heart failure (15.1% versus 16.2%, respectively, $p = 0.87$, data not shown) (2). Since these conditions show collinearity and most previous studies exploring aspirin resistance were performed in patients with ischemic heart disease, we just included the presence of coronary heart disease as a potential predictor of aspirin resistance (3). Among other suggested inflammatory conditions suggested, none of our patients had a previous history of stroke; only one diabetic male suffered from a primary Reynaud phenomenon, which was not associated with any connective tissue disease.

Serum levels of more sensitive and certain experimental inflammatory markers, such as interleukin-6 or high-sensitivity C-reactive protein, and markers of oxidative stress may have a relationship with aspirin resistance. However, we presume that the clinically significant effect size of the aforementioned factors on aspirin resistance would be small, if any, due to the presence of low numbers of patients with so-called inflammation (i.e. coronary heart disease) in our study. Yet, Yalcinkaya and Celik are right about the impact of inflammation on aspirin resistance, since the inflammation is closely related with platelet activation.

Finally, we conducted this study to explore the effects of overt diabetes mellitus (DM) instead of insulin resistance on aspirin resistance. Perhaps the comparable frequency of insulin resistance resulted in a comparable frequency of aspirin resistance in both groups. No matter what, our primary interest was the relationship, not the causality. Demonstration of such an effect of insulin resistance instead of DM on aspirin resistance nevertheless would not change our conclusion.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

1. Yalcinkaya E, Celik M. Evaluation of inflammatory conditions associated with aspirin resistance. Ups J Med Sci. 2014; Epub ahead of print.
2. Tasdemir E, Toptas T, Demir C, Esen R, Atmaca M. Aspirin resistance in patients with type II diabetes mellitus. Ups J Med Sci. 2014;119:25–31.
3. Grove EL, Hvas AM, Johnsen HL, Hedegaard SS, Pedersen SB, Mortensen J, et al. A comparison of platelet function tests and thromboxane metabolites to evaluate aspirin response in healthy individuals and patients with coronary artery disease. Thromb Haemost. 2010;103:1245–53.

Correspondence: Tayfur Toptas, Van Bolge Egitim ve Arastirma Hastanesi Hematoloji Poliklinigi, Kat 1, Van, Turkey. E-mail: toptast@gmail.com

(Received 15 March 2014; accepted 22 April 2014)

ISSN 0300-9734 print/ISSN 2000-1967 online
DOI: 10.3109/03009734.2014.918680